## **AMENDMENTS**

## **Listing of Claims**

The following listing of claims replaces all previous listings or versions thereof:

## 1-43. (Canceled)

- 44. (Currently amended) A method of assessing protein stability, folding and/or solubility comprising:
  - a) expressing in a host cell a fusion protein comprising (i) a protein of interest and (ii) a first segment of a marker protein, wherein said first segment has only systemicsystematic effects on the stability, folding and/or solubility of the protein of interest;
  - b) contacting said fusion protein produced in step a) with a second segment of said marker protein, wherein said second segment is capable of structural complementation with said first segment; and
  - c) determining structural complementation,

wherein a greater degree of structural complementation, as compared to structural complementation observed with appropriate negative controls, indicates stability, proper folding and/or solubility of said protein of interest.

- 45. (Previously presented) The method of claim 44, wherein said fusion is C-terminal to said protein of interest.
- 46. (Previously presented) The method of claim 44, wherein said fusion is N-terminal to said protein of interest.
- 47. (Previously presented) The method of claim 44, wherein said marker protein is selected from the group consisting of a target binding protein, an enzyme, a protein inhibitor, a fluorophore and a chromophore.

- 48. (Previously presented) The method of claim 47, wherein said marker protein is a target binding protein.
- 49. (Previously presented) The method of claim 48, wherein said target binding protein is ubiquitin.
- 50. (Currently amended) The method of claim 47, wherein said marker protein [[is]] comprises a chromophore.
- 51. (Currently amended) The method of claim 50, wherein said ehromophoremarker protein is green fluorescent protein, blue fluorescent protein, yellow fluorescent protein, luciferase or aquorin.
- 52. (Previously presented) The method of claim 47, wherein said marker protein is an enzyme.
- 53. (Currently amended) The method of claim 52, wherein said enzyme is β-galactosidase, eytochrome c, chymotrypsin inhibitor, luciferase, Rnase, phosphoglycerate kinase, invertase, staphylococcal nuclease, thioredoxin C, lactose permease, amino acyl tRNA synthase, and or dihydrofolate reductase.
- 54. (Previously presented) The method of claim 53, wherein said enzyme is  $\beta$ -galactosidase.
- 55. (Previously presented) The method of claim 54, wherein said first segment is the  $\alpha$ peptide of  $\beta$ -galactosidase, and said second segment is the  $\omega$ -peptide of  $\beta$ -galactosidase.
- 56. (Currently amended) The method of claim 44, wherein said protein of interest is Alzheimer's amyloid peptide (Aβ), SOD1, presenillinpresenilin 1 andor 2, α-synuclein, amyloid A, amyloid P, CFTR, transthyretin, amylin, lysozyme, gelsolin, p53, rhodopsin, insulin, insulin receptor, fibrillin, α-ketoacid dehydrogenase, collagen, keratin, PRNP,

immunoglobulin light chain, atrial natriuretic peptide, seminal vesicle exocrine protein, β2-microglobulin, PrP, precalcitonin, ataxin 1, ataxin 2, ataxin 3, ataxin 6, ataxin 7, huntingtin, androgen receptor, CREB-binding protein, dentaorubral pallidoluysian atrophy-associated protein, maltose-binding protein, ABC transporter, glutathione S transferase, andor thioredoxin.

- 57. (Previously presented) The method of claim 44, wherein said negative control utilizes a fusion protein that is improperly folded and/or insoluble.
- 58. (New) The method of claim 47, wherein said marker protein is cytochrome C or chymotrypsin inhibitor.